Amendments to the Specification

At page 26, please amend the paragraph beginning at line 9 as follows:

More importantly, the addition of exogenous uridine did not significantly reconstitute infectious virus production in A771726-treated, CMV-infected EC (results shown in Figure [[3B]] 2). Thus, A771726-mediated inhibition of CMV activity appears to be independent of the inhibitory effects of this agent upon pyNTP synthesis.

At page 30, please amend the paragraph beginning at line 10 as follows:

Results representative of 2 replicate experiments are presented in Figure [[5]] 3. IE1 and gB band densities (as determined by scanning densitometry) are plotted as ratios to those of GAPDH. As demonstrated in this figure, while gB mRNA expression was completely suppressed in cells treated with PFA/GCV, A771726 exerts no inhibitory effect upon transcription of either IE1 or gB genes

At page 32, line 28 through page 33, line 11, please amend the paragraph as follows:

While uninfected cell extracts inhibited no detectable enzyme activity, extracts derived from CMV-infected cells assayed in the absence of A771726 or PFA exhibited specific viral DNA polymerase activities in the range of 276-379 nM incorporation/hr/mg protein. Representative results of experiments performed with extracts of VHL/E-infected HUVEC or P8-infected HFF (2-4 replicate experiments per virus/cell combination) are presented in Figure [[6]] 4. Data shown in Figure [[6A]] 5, (specific enzyme activity expressed as percent of PFA/A771726-free controls), demonstrate that, while PFA reduced viral DNA polymerase activity in a concentration-dependent manner (with complete inhibition at 1 mM). A771726 showed no detectable inhibitory activity even at concentrations which dramatically reduced plaque formation (Figure [[6B]] 5). Experiments performed with extracts prepared from CMV strain BUR/E-infected HUVEC generated essential identical results.

At page 34, line 19, please amend the paragraph starting at line 19 as follows:

All current anti-CMV chemotherapies focus upon inhibition of viral DNA replication, although specific mechanisms vary slightly among different agents. Clinical strains have emerged which exhibit cross-resistance to multiple drugs. The effect of leflunomide product on activity of drug-resistant virus was evaluated as follows. CMV strain D16 was isolated from the same patient as strain P8. However, unlike P8, D16 exhibits multi-drug resistance. Plaque reduction assays performed in HFF cultures as described above (results shown in Figure [[8]] 7) revealed equivalent sensitivity of these two isolates to A771726 (ID_{50} ~40-60 μM). Thus leflunomide-mediated inhibition of viral activity occurs independent of resistance to current clinical chemotherapeutic agents.